

A. Thesis for Graduation.

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T H E . T O N S I L S . in health , and disease.

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Until quite recently the Tonsils were considered to be useless structures, or were the functionless survival of some pre-existing organ and there have been many theories propounded as to their uses , and their presence was considered of so little importance that even some of the modern text Books of Physiology and Histology, as those of Bennett, Foster, Mc Kendrick, and Carpenter, do not condescend to notice them; others again refer to them briefly, as Flint, (in his text book) observes" that they are an aggregation of from ten to "twenty compound follicular glands, held together by "fibrous tissue, and that they secrete a greyish viscid "mucus containing a number of leucocytes and disquamated epithelial scales .

Within the last few years however our knowledge of these organs has considerably increased by studying their structure and development both in the human subject, other mammals, and the bird frog and salamander, and we now more readily understand their presence and function, namely:-to produce leucocytes, which perform their work as "phagocytes"- in fact the Tonsils are protective organs . To have arrived at this definite conclusion it will be necessary to consider

their development and structure both in the human subject and in animals, but before doing so it will be better to recognise definitely the position of these protective organs.

They are four in number and consist of the two faucial tonsils, the pharyngeal tonsil, the lingual tonsil and the adenoid infiltration described by Bickel (4) and Stohr (5).

The faucial tonsils are those which are commonly known to us situated in the glosso-epiglottidean hollows between the anterior and posterior pillars of the fauces .

The Pharyngeal tonsil or Tonsil of Luschka, lies on the upper part of the posterior wall and roof of the naso pharynx .

The lingual or fourth Tonsil, which is situated at the base of the tongue between the circumvallate papillae and the epiglottis .

The Adenoid Infiltration of Bickel (4) and Stohr (5), which runs along the floor of the nose and upper and lower surface of the velum palati .

The Tonsils with the Adenoid Infiltration form two complete rings ; a lower one formed by the faucial and lingual tonsils with the adenoid infiltration on the under surface of the velum palati, and an upper

ring formed by the pharyngeal tonsil and infiltration running along the floor of the Nose and upper surface of the velum palati . The lower zone of adenoid tissue forms a barrier between the pharynx and mouth of "protective" tissue ,and the upper a "protective "barrier "between pharynx and nose .

#### DEVELOPMENT:

They appear in the four month of intra uterine life in the human embryo in the form of a little fissure on each side, on a line with or above the opening of the eustachian tube . In the fifth month each tonsil is a flat ~~sae~~ with a slit like opening<sup>I</sup> ; very like the tonsils which are seen in a Frog ,single shallow pits (Holl.3.). The outer wall is thickened and contains numerous leucocytes ,which are not however contained in separate follicles . At a later period the follicles are formed by the growth of connective tissue septa . The tissue elements proceed both from the epiblast and the mesoblast ,the former supplying the cellular elements; the latter the connective tissue septa, the blood vessels and probably also the lymphatics whilst in most glands the basilar layer of the epithelium forms the whole of the involution ; according to Retterer(2) in the tonsils the whole thickness of the epithelium investment proliferates . The epithelium, as elsewhere, in the mouth being



stratified squamous .

Gulland(6) does not agree with Retterer, who he says, has made a fatal mistake in trying to prove that Tonsils are true Glands - his idea being that the germ centres are really glandular follicles and are developed from "epithelial buds" which are met with in the developing tonsil. These are really sections which do not reach the lumen through the epithelium of crypt nor does he seem to realise the presence of leucocytes at all. His. (4) Bickel and Retterer are all agreed as to the importance of the epithelial folds in the primary production of the Tonsils . To explain the reason of the occurrence of the epithelial fold in the first place is a difficult matter, and various theories, as those of Schwabach(8), have been advanced, which will not hold water . Gulland's(6) observations go to prove that the "connective tissue" plays an almost entirely passive part in the development of the Tonsil ; the active role is performed by the epithelium . The ingrowth of a blunt wedge of epithelium, as it were, condenses the connective tissue into which it passes; which connective tissue is already dense<sup>↑</sup> . The wedge pushes itself at right angles to the circular fibres of the connective tissue and by the slight irritative effect which it must have - likening it to the grafting of a foreign body to connective tissue - new blood vessels are formed and

there is the usual proliferation of connective tissue cells". This process begins before the advent of the leucocytes, for the embryo is several days old before these appear in the blood (Gulland.11), and can thus be more clearly seen; but it does not stop there. The large number of leucocytes, which are massed together, he explains, by a chronic vascular dilation having occurred owing to this constant irritation and that the blood vessel walls become permanently permeable; but is this supposed irritation described by Gulland, consistent with nature? Can there physiologically be a constant irritation, even on a small scale, going on in the walls of pharynx?.

At term the Tonsils are fully formed.

Schwabach(8) describes the epithelium over the upper and anterior part of the pharyngeal tonsil as generally ciliated. The epithelium of the tonsils however differs considerably from that of the rest of the mouth and pharynx, as its continuity is broken by the emigration through it of large numbers of leucocytes. In all the tonsils the leucocyte infiltration appears first near primary pit.

#### HISTOLOGY :-

1. A supporting framework or reticulum of connective tissue in which are blood vessels & lymphatics, and packed together numerous cells, lymphocytes, (Gulland.11).

Hodenphyl(9) describes two forms of tissue ;one which he prefers to call reticular tissue,in which there are no connective tissue cells and which is found in between the nodules or "germ centres",and a finer variety which is formed in the nodules or "germ centres" in which the meshes are lined with endothelium . In the 'germ centre'the reticulum is disposed irregularly, but at its margin is condensed,and the fibres are so drawn out that the meshes are at right angles to the radius of the sphere of the germ-centre and the fibres form a sort of capsule to it produced by centrifugal pressure ; outside the capsule the fibres become continuous with those of the ordinary adenoid tissue.

## II. Blood Vessels:-

The vessels which supply the faucial tonsils in particular,are the :-

- I. Branches from the dorsal artery of the tongue.
- II. " " " Inferior palatine.
- III. " " " superior palatine .

The larger trunks after entering the tonsil are surrounded by fibrous tissue,developed from the basal capsule; they are short, almost immediately breaking up into capillary vessels .

The veins are very numerous, of large size and have very thin walls,and are in connection with branches of the internal jugular .

111. Lymphatics.

The lymphatic vessels commence in the Tonsils by open radicles in the connective tissue spaces and join into several trunks just as they leave the Tonsil which penetrate the basal capsule and connect with the lymphatics at the base of the Tongue, which in turn enter the lymph glands at the angle of the jaw about the level of the hyoid bone . It is to be noted that that there are no lymphatic vessels inside the germ centres, nor where germ centres underlie epithelium are there any on the epithelial side of the germ centre, but there are numerous lymphatic vessels just outside the capsule ready to receive any cells which pass through it. There are no lymphatic vessels passing to the Tonsil, (Gulland).

Walker Downie (36) in a Paper read before the Glasgow Medical-Chir Society, November 1895; says with regard to the faucial Tonsils:- "They are isolated and though they have no large afferent vessel such as the typical lymph gland possesses, there are numerous small lymphatic vessels which gather in the fluids absorbed by the buccal & faucial mucous membrane in the immediate neighborhood of the Tonsil and these may be considered to take the place of the main vessel of the typical lymph gland" and "although lymphatics communicating with large lymphatic spaces close by exist in the neighbourhood of the faucial Tonsils, yet



no connection has yet been traced between these lymphatics or the lymph spaces and the tonsils and their follicles . Thus here are two entirely opposed statements - Gullands' published in 1891 and Walker Downies' in 1895. The latter I think I may say evidently has not gone very far into the function of the tonsil, as he describes, further on in his paper, the tonsil as having "a somewhat viscid secretion, a fluid which is endowed with highly amylolytic properties and has certainly not been acquainted with the observations of Gulland (10), Hosenphyl (24), Dmochowski (37), Dienlafoy (38) and others, as we shall see later on .

#### IV. Nerves:-

No definite nerve supply to the tonsil has, as yet, been demonstrated.

V. Germ centres and Cells:; or as they are usually called in this Country, follicles; have the appearance on section (after being stained with Flemings fixative) of round clear spots surrounded by a dark ring and are arranged radially round the crypts . Gulland (10) thinks that the prime cause in their formation is to be found in the blood vessels . They have a peculiar arrangement of blood vessels; only capillaries enter the germ centre and these pass in, in a radially centripetal manner, curve on themselves in the centre, and pass out again to enter the venule ; this results in the

massing of terminal capillary loops at the centre, and therefore, to quote Gulland(10)"while the current of blood in the afferent capillaries is radially centripetal, the pressure outside the capillaries, produced by the vis a tergo of exudation will be radially centrifugal". The blood supply to any cells in the germ centre will thus be very abundant.

The germ centres are part of the tonsil specially differentiated in order to forward the mitotic division of leucocytes. Leucocytes multiply only by mitosis- or indirect division- and this mitosis takes place only in adenoid tissue, mainly the germ centres of tonsils, spleen, lymph glands &c.

Thus any lymphocytes which pass out of the capillaries in the germ centre will be held for some time in the fine meshes of the connective tissue in the interior, already referred to, but will eventually be pushed gradually either into a lymphatic vessel or into the general adenoid mass of the tonsil. In the latter situation the leucocytes are mainly of the hyaline variety, relatively stationary cells and one is able to trace every gradation from the lymphocytes to the large macrophages which lie under the epithelium. On the other hand with regard to the cells passing through the epithelium to the crypts, it will be found that those cells passing from the side of a germ centre, nearest

the crypt, where the epithelium has been almost entirely removed by the action of the leucocytes and consequently where the passage is easy, are almost all lymphocytes or hyaline cells, but whenever the epithelium is largely intact, so that its passage involves movement on the part of the leucocytes themselves, those underlying the epithelium are hyaline, and Gulland(11) says that as they pass through the epithelium the cells and nuclei lengthen and assume whatever shape is necessary and when they reach the surface the nuclei are polymorphous and the cells absolutely indistinguishable from the oxyphile leucocytes of the blood. They have in fact adopted the form most suitable to their environment.

Those which have remained stationary in the adenoid tissue, where they are richly supplied with blood, have retained their original character as on starting, namely:- hyaline; those which have left the tonsil proper and have had difficulty in so doing, have undergone changes in nucleus and cell body.

The lymphocytes on the other hand which have passed from the germ centres of the tonsil into lymph vessels are carried with the lymph into the blood; some are arrested for a time (or permanently) in the lymph glands where they may become more highly specialised.

The greater number however are stationary cells, probably owing to the very free blood supply.

The pale appearance of the germ centre (with nuclear

stain) on section is due to the fact that most of the leucocytes contained in it are principally large resting cells about to divide . The dark ring is caused by the heaped up lympho-cytes in great numbers at the periphery of the germ centre .

Those leucocytes which emigrate through the channels in the epithelium or through the individual cells of the epithelium (Stohr, 12) enter the lumen of the crypt and thus pass into the cavity of the pharynx and the mouth . Hodenphyl (9) has adopted the views of Zawarykin (13) who calls it a process of "rarefaction of the epithelium" the results of a pressure beneath upon the epithelial lining and Hodenphyl ascribes to this process the great irregularity in the contour and thickness of the epithelial layer . The leucocytes however actually destroy the epithelial cells and it will be found that the destruction is most nearly complete , and the passages through the epithelium are widest , just above the germ centres where the constant attack of fresh leucocytes prevents the repair of the epithelial covering - (Gulland (10) and the theory of Hodenphyl and Zawarykin, that this emigration is caused by a process of "rarefaction of the epithelium" is an entirely erroneous one . This emigration is a normal occurrence and takes place almost always whenever there is an accumulation of leucocytes underlying



epithelium, as in the small intestine, nasal mucous membrane and urinary bladder &c (Stohr).

The epithelium of the pharyngeal tonsil differs somewhat from that of the faucial .

In the first place it is ciliated and dips down into the recesses and crypts - which is continuous with that lining the respiratory part of the interior of the nose and adjacent mucous membrane of the nasopharynx.

The epithelium consist of more than one layer of Cells; the layer of ciliated cells, with the deeper cells forming two or three layers which rest upon a well-defined basement membrane .

Before going further to the consideration of the functions of the tonsils it will be as well to dwell for a little time on the varieties of the leucocytes , and one will thus be able to follow better the functions of Tonsils in health and disease.

In the first place what is a leucocyte? :- a  
UNICELLULAR ORGANISM WHICH IN THE MIDST OF vertebrate

tissues retains the character and habits of a Protozoon.  
 (Gulland (14)). They have the power and character  
 of being able to wander from place to place by  
 amoeboid movement; they are carried about from one  
 adenoid tissue to another by the blood vessels and  
 lymphatics .

Secondly, they have the power of ingesting  
 foreign materials ,as micro-organisms and dead  
 tissues and of utilising these particles ,when they  
 are capable of being so ,for nutrition(Metschinkoff,  
 (15)).

Thirdly, the form of leucocytes vary very much  
 (Muller, (16) but they are all stages in the life  
 history of an unicellular organism .(Gulland. (14)).

There have been various methods of classifying  
 leucocytes ,as by their size,nucleus,or granules,  
 (Ehrlich(17)). Leucocytes as we have said  
 already ,multiply only by mitosis or indirect  
 division, though other theories have been -

advanced by ,~~Dreus~~(18) ~~Stohr~~(12) and others; and it is from the daughter cells or lymphocytes that we can best trace their process of development .

These young leucocytes or lymphocytes as we have already seen are heaped up in great numbers at the periphery of the germ-centre; they possess little power of amoeboid movement. They pass into the lymphatics and are to be found in the channels of the epithelium of the Tonsils .

The lymphocytes grow when they are able to get sufficient nutriment and form the next variety of leucocyte . Simply a leucocyte which has grown a little, through extra nutriment and therefore to be found where the blood supply is good, as in lymphatic glands and between the germ centres .

Ruffer(19) calls these cells when found in the intestinal wall, containing micro-organisms:-microphages.

LEUCOCYTES now divide into two varieties:- the Stationary and the wandering.

Stationary, or Phagocytes, of Metschnikoff, or Macrophages of Ruffer, are lymphocytes which having found themselves in favourable circumstances grow enormously. They do not always remain stationary, for if the tissue they are resting in is starved they may move a certain distance for nutriment. Heidenhain(20). these large stationary cells are capable of ingesting various materials, as micro-organisms, carbon and dust particles,

red blood corpuscles, pigment, leucocytes in various stages of degeneration and decay . They are found situated in the human tonsil in considerable numbers just below the epithelium .

Wandering leucocytes, are very different from the stationary cells . They are also derived from the lymphocytes, which have wandered off into the lymph stream into the blood or into the tissues surrounding the tonsil or other lymphatic organ . They may have one or more nuclei. They are found in great numbers where emigration from the blood vessels is taking place, in granulation tissue or irritated connective tissue and Gulland(14) maintains that the more difficult it is for the leucocyte to force a passage the more will its nucleus be elongated or ramified .

There seems to be some confusion over the terms, stationary and wandering cells, as Gulland.(14) calls Ruffer's<sup>†</sup>(19) "Macrophages", stationary cells, whilst Ruffer himself calls them wandering cells .

The reason of this as far as we can make out is that the stationary cells of Gulland are only relatively so, as they must have the power of movement, as it is only by throwing out pseudopodia that they can ingest foreign particles which form their food. What Gulland means by stationary is that they confine their wanderings to a very small range .



We are now in a position to study the

### FUNCTIONS OF THE TONSILS :

Not so very long ago the Tonsils were supposed to be functionless, or to be the remnants of some pre-existing Organ . Now we know that in mammals , especially the primates, they are highly specialised when we compare them with those of the Frog, (2) Salamander &c.

The older theories will not require very much consideration, as most of them have no scientific basis whatever. Such as; their presence in the foetus was to prevent the meconium from passing into the pharynx etc; that they had some relation to the generative system and even now, in practice, one comes across some fond parent who objects to his Son's Tonsils being removed for hypertrophy, on the Ground that it will cause atrophy of the Testicles .

Another theory was that the tonsils are secreting organs, - secreting a thick viscid mucus which lubricates the bolus of food and the posterior part of the mouth and pharynx (Quain's Dictionary of Medicine (25)).

Undoubtedly there are a few mucous glands in the covering of the tonsil, but not more than in the adjacent mucous membrane of the mouth .

Again, Perrin imagined that they were concerned in voice production, also that they were compensatory

organs for warming inspired air when there is nasal obstruction .

The more recent theories are those of Hill(21), Scanes Spicer(22), Hingston Fox(23), Hodenphyl(24), Gulland(10) and Walker Downie(36) and of these , Gullands is the only one which, with our present knowledge, can be entertained .

Gulland and Hodenphyl have discussed the theories of Hill , Spicer and Hingston Fox .

Hill(21) imagined the tonsils to be absorbent organs and thinks, that they have to deal specially with the products of salivary digestion, which are absorbed by the buccal and faucial mucous membrane and passed thence by small lymphatics to the tonsils, but salivary digestion is simply the conversion of starch into maltose by the ptyalin ferment in the salivary secretion and is not a true digestion, and what is more, only a portion of the starch is converted, the food usually not remaining long enough in the mouth ; besides if Hill had examined the pharynx of the carnivora he would never have propounded this theory, as they have large tonsils, and their food does not contain starch, and we have seen that there are no lymphatics passing to the tonsils for as Gulland(10) points out "the tonsils are peripheral lymph glands and not intermediate"

Scanes Spicer (22) in a somewhat lengthy article brings forward a theory somewhat similar , viz:-that

the tonsils are concerned in absorption and that each tonsil, faucial, <sup>n</sup>lingual, and pharyngeal absorbs from the nose and mouth respectively . He says ,it is a general rule that fluids thrown out in the intestinal canal are absorbed by the segment of intestine below, and considers, along with Hingston Fix(23) that the same holds good with the tonsils, that they act in the prevention of fluid waste in the economy, by re-absorbing the buccal secretion to a large extent after their work is done . Secondly that they absorb certain elements of the food bolus as it is squeezed past them in deglutition. This theory can be dispensed with as the contact of the food bolus with the tonsils is only momentary; and thirdly that they form part of the bloodmanufacturing system acting, as Hingston Fox expresses it, as "nurseries for young leucocytes, planted "by the waterside, and drawing their sustenance from "the nutrient stream". Unfortunately for Hingston Fox's theory the young leucocytes are unable to draw sustenance, for as we have before mentioned the Tonsils are peripheral and not intermediate lymph glands .

To the Pharyngeal Tonsil, he ascribes the same functions, viz:-the prevention of waste of some of the nasal secretion . He claims that chronic nasal catarrh is the cause of hypertrophy of this tonsil and would clinch his argument thus:-"the foulness of "the secretions which are dislodged in digital

"examinations of post nasal growths keep up the  
 "enlargment commenced by an ordinary catarrh",but is  
 not the presence of these large pharyngeal growths  
 sufficient per se to keep pent up the normal secretion  
 and being pent up and becoming contaminated is septic?

Passing to the Lingual Tonsil, Spicer along with  
 Dobell states a most extraordinary theory,viz:- that  
 the uvula in the erect posture serves as a guide for  
 secretions to run down from the naso pharynx and land  
 on the lingual tonsil,where they are absorbed. In  
 the first place we do not think that normally the uvula  
 has so much moisture about it that it can ever be  
 considered to drip. During the last few months we  
 have not among the numerous throats we have examined  
 both in Hospital and private practice ,been able to  
 discover the uvula in that state of moisture described  
 by Spicer(25). Again we know what happens when we  
 get the uvula in what is called a relaxed condition-  
 irritation of the epiglottis and larynx and tickling  
 cough,and granting that his theory was correct,every  
 time we moved our heads out of the erect posture ,a  
 drop of this nasal secretion would fall in<sup>to</sup> the larynx.,  
 as the uvula would not drip"in a plane anterior to the  
 "epiglottis"as described by Spicer . We may therefore  
 leave Spicers'views and briefly look at those of  
Hingston Fox:-



He emphasises the fact that the food bolus is brought into contact with the Tonsils- indeed "it must be actually rubbed against their surface during its passage"; but again we point out that this must be only a momentary operation . He thinks that some of the food is pressed into the tonsillar crypts- but Gulland (10) points out that while some the crypts in the human tonsil may open towards the mouth, that in the rabbit, cat, and guinea pig, the single crypt opens towards the posterior aspect <sup>which</sup> ~~of~~ is covered anteriorly by a lip.

Fox goes on to say that an interchange takes place between the glands and the food and that from the structure of the glands ~~the~~ir function is evidently to absorb and he compares them with the solitary and agminated glands and brings forward;

"the observations of Schaefer (26.27) on the absorption of fat particles, by the amoeboid cells of these glands, also those of Stohr (12) to which we have already referred.

He regards the adenoid tissue of the tonsils as the birth-place of leucocytes and then closes with his poetical simile" that the tonsils are nurseries of young "leucocytes planted here, as it were by the riverside, "and drawing their sustenance from the nutrient stream", to which we have alluded and laid aside as impracticable, as the tonsils have no lymphatics going to them.

Hodenphyl (24) endeavoured to ascertain the amount

of absorption which may take place in the tonsils .  
 The experiments were done on dogs and rabbits . He tried  
 the rubbing in of olive oil, melted lard, lanolin and  
 on killing the animals fifteen minutes afterwards he  
 found on examining the tonsils microscopically , a  
 few fat droplets generally near the surface (they  
 seemed to be between the cells). He then tried carmine,  
 berlin blue, emery powder suspended in water, also  
 aniline dyes , with negative results .

He came to the following conclusions:-

- I. Soluble or insoluble materials are not absorbed by  
 the tonsils except to a very slight extent .
- II. It is the epithelial covering which prevents the  
 absorption.
- III Whatever the function of the Tonsil is, it is  
 certainly not concerned with absorption.

With regard to Walker Downies' (36) views which  
 were read before the Glasgow Medical Society in November  
 1895 and therefore after Gulland and Hodephyl had  
 published theirs, it would appear that he has not seen  
 either paper or if so entirely ignores their conclusions,  
 and makes some astonishing statements , giving no  
 authority for them .

- I. There is the statement that there are a number of  
 lymphatics, small in size passing to the faucial  
 tonsils, which statement is entirely opposed to that

of Gulland. He appears evidently to have mistaken the channels and roadways made by the leucocytes through the epithelium, for small lymphatics passing to the tonsil.

2. His second statement is that there are no efferent lymphatics passing from the faucial tonsils, and wonders at it. Now Vesalins in 1543, Morgagni in 1765, Kolliker in 1852, Rosenberg in 1892 and others maintain that there is a direct communication with all the large lymph spaces in the neighborhood which in turn connect with the deep cervical glands and Woodhead(28) bears this out from patholigical experience.

3. Thirdly he talks of the "tonsillar secretion" as a "somewhat viscid fluid which is endowed with highly "amylolytic properties" mixing with" and very possibly "aiding in the digestion of the bolus and going with "it into the stomach". The Tonsils have been proved now to have no secreting cells(Gulland, Fox, Spicer &c), certainly none that have "amylolytic properties " and within a few words of the above remarks he makes the tonsils absorb "some of the products of salivary "digestion, as well as excessive buccal, nasal and "pharyngcal secretions". In his opinion therefore the tonsils both secrete and absorb a salivary secretion which we have proved is impossible.

Lastly we come to the views of Gulland(10) which are the most practicable . He came to the following conclusions:-

- I The Tonsils, (faucial lingual and pharyngeal) are organs arranged to further the reproduction of leucocytes. c
- II. This reproduction takes place mainly in the germ centres by mitotic division of pre-existing leucocytes.
- III. The young leucocytes so formed are partly carried off to the general circulation by lymphatic vessels originating in the tonsil; partly remain in the tonsil as stationary cells and partly wander out into the crypts by perforating <sup>through</sup> ~~into~~ the epithelium .
- IV. They thence pass to the surface of the tonsils and take up foreign bodies , especially micro-organisms which would otherwise pass the tonsils .
- V. The tonsils in the human subject form a protective ring both for the Alimentary and respiratory tract.
- VI. There is no reason to regard the tonsils as having any absorbent function in normal circumstances ; the reproduction of leucocytes is sufficiently active as a rule to keep up a continuous outward stream of these cells and to prevent the entry of foreign substances into the tonsils .

Now let us see how Gulland comes to the conclusions:-



1. That the Tonsils are organs arranged to further the production of leucocytes and

11. That this production takes place mainly in the germ centres, and that the young leucocytes are carried, some into the circulation; others remain stationary cells in the tonsil, and others wander out into the crypts through the epithelium.

We have seen the peculiar arrangement of blood vessels in the germ centres, viz:- that the current of blood in the afferent capillaries is centripetal and the pressure outside the capillaries is radially centrifugal.

11. That the germ centres are arranged radially round the tonsillar crypts and that seen on section (with nuclear stain) they are pale in the centre which is principally due to large resting cells about to divide, and that that dark outer ring is due to heaped up leucocytes (lymphocytes) in great numbers at the periphery, which have just divided by mitosis, or indirect division.

111. We have also seen that the majority of young leucocytes pass, on the one side into the lymphatic system, some remain stationary cells, others pass through the epithelium to the tonsillar crypts (Stohr (5)).

IV. That the leucocytes-or as we may more popularly call them, phagocytes- pass to the surface of the Tonsils and take up foreign bodies especially micro-organisms

which would otherwise pass the tonsils .

Ruffer(19)found in leucocytes,near the free surface of the dogs tonsil,masses of carbon and extraneous substance ,and in the rabbit a much less quantity,and he attributes this to the rabbit being a very much cleaner eater than the Dog ,who goes roaming about the Street poking his nose into all kinds of dirt and refuse; also that the Dogs tonsils are relatively much larger than the rabbits- to this we shall refer again later.

Also that on staining sections of the tonsil of the dog and rabbit with Loefflers' blue or gentian violet some of the macrophages are found to contain dead leucocytes and numbers of micro-organisms ,generally micrococci,though baccili are found . Both Ruffer(19) and Metschinkoff(15)on examining the mucous covering the surface of the tonsils ,found in it numerous leucocytes crammed with micro-organisms,and Woodhead(28) says it might be possible in the case of motile bacteria to assume that,in the process of their passage into a leucocyte they play an active part and the leucocyte merely a passive <sup>A</sup>role,but in the case of the non-motile organisms of which the tubercle bacillus is only a single example ,it is difficult to understand how they can find their way into leucocytes unless these latter become the active agents in the process. Pathogenic organisms are found constantly even in the mouths of

healthy subjects . Staphylococci have been isolated from the normal saliva by many observers ,also the pneumococcus ,whilst Löffler(29) cultivated the bacillus diphtheriae from a healthy child's mouth .

These micro-organisms must of course pass over the Tonsils, but if they try and penetrate the tissues of that organ they are at once seized and destroyed by the micro, and macrophages .

Ruffer(19) maintains that there is a constant warfare going on, in (more probably on) the surface of the tonsil between the micro-organisms and the amoeboid cells, for from all these tonsillar crypts , leucocytes are constantly passing out , so that it would be difficult for the micro-organisms abounding in the mouth to pass back into the pharynx without coming in contact with them, (Miller(30)). Gulland(10) says he found great difficulty in finding micro-organisms in the stationary leucocytes (Macrophages of Ruffer) lying just under the epithelial lining of the crypts .

Conclusion No 5 is to the effect that the lingual and faucial tonsils form a protective ring, between the mouth and the rest of the digestive tract and that the pharyngeal tonsil with the diffuse infiltration on the floor of the nose and upper surface of the velum palati form a protective ring shutting off the nose from the respiratory tract .

When studying the anatomical relations of the various tonsils we found that they formed two distinct zones, one between the pharynx and the mouth consisting of the faucial tonsils, the lingual tonsil, and the adenoid tissue on the lower surface of the velum palati, when the pharynx is opened from behind and the mouth closed it will be observed that the posterior pillars of the fauces lie against the tongue surface, as do those in front; the tonsils situated on each side in the interval between the pillars lie also against the tongue, resting upon the lingual tonsil; and are separated one from another by the uvula, and the cavity of the mouth is thus shut off from the pharynx.

The other zone we have seen consist of the pharyngeal tonsil and the adenoid infiltration on the floor of the nose and upper surface of the velum palati. Weidersheim, referred to by Killian (34) suggests that where animals with wide nostrils (e.g. Man and domestic animals) live much indoors in close rooms where there are many corpuscular elements in the air, we might expect the pharyngeal tonsil to be large, whilst in cases where the nose is complicated in form, so that the air is purified, or where the stream of air does not fall directly on the pharyngeal tonsil, that organ would not be formed at all, or would at best be small, as in the case of the Horse.



Gulland does not imply that these adenoid structures form an absolute barrier between the mouth and the Alimentary canal, on the one hand and the nose and the upper pharynx on the other, but that they are only part of a very extensive set of protective appliances; - for instance in the mouth various authorities state that the healthy buccal saliva has a definite bactericidal power.<sup>1</sup> Also St Clair Thomson and Hewlett (31) have studied very minutely "the fate of micro-organisms in "the inspired air". They calculated that normally about 1500 organisms are inhaled into the Nose every hour and in an atmosphere, such as that of London, as many as 14,000 . Gunning (32) in 1882 showed that expired air contained no microbes capable of provoking putrefaction in sterile liquids through which it had passed and in 1887 Strauss and Dubreuil (33) arrived independently at a similar conclusion .

St Clair Thomson and Hewlett came to the following conclusions, that nearly all the micro-organisms of the air are arrested before reaching the naso-pharynx ; probably the majority are stopped by the vibrissae at the very entrance of the nose and those which do penetrate as far as the mucous membrane are rapidly eliminated . The nasal mucous membrane is an unsuitable soil for the growth of micro-organisms and hence is an important factor, in that it does not further their multiplication.

<sup>1</sup> Hugenschmidt (87)

The removal of the intruding organisms from the schneiderian membrane is probably in the main due to the action of the ciliated epithelium ,assisted by the trickling of mucus and the lachryncal secretion. Phagocytosis may share in the work of removal ,though to a small extent,for we have only once found phagocytic cells containing bacteria .

The pharyngeal tonsil and adenoid infiltration , then in health according to these observers,have not very much to do ,as they consider that most of the work of protection is carried on by the ciliated epithelium &c and they have only once found phagocytes containing bacteria . Wartz and Lermoyez (35)held that the nasal mucus itself exerts a bactericidal influence in all or nearly all pathogenic agents, St Clair Thomson and Hewlett were so far however not able to corroborate their researches .

The Tonsils then we must consider are only part of a great mechanism to arrest the entrance in the respiratory tract and alimentary canal of micro-organisms in the air which we breath ,and the food we eat .

VI. And lastly that there is no reason to regard the tonsils as having any absorbent function in normal circumstances .

We have discussed the observations and experiments of Hodenphyl who proved conclusively that whatever was

the function of the tonsil it was not that of absorption and we have discarded the views of Spicer and Hingston Fox as impracticable having no physiological or physical basis for we have Gullands' definite statement that there are no lymphatics going to the Tonsil ,that it is a peripheral and not an intermediate lymph organ.

We have seen that in the healthy tonsil there must be a continual passage of leucocytes through the epithelium into the crypts and that there is thus no means of entrance for micro-organisms ,as the stream of leucocytes in an opposite direction will prevent their entrance .

We have now discussed the functions of the Tonsils and may adopt the views of Gulland,viz:-that the function of the tonsils is to produce leucocytes and to aid in a great scheme of protection to the alimentary and respiratory tracts by a process very much allied to Metschinkoffs(phagocytosis carried on by the leucocytes of the circulation .

# PATHOLOGICAL CHANGES occurring in the TONSILS

--and their bearing on Disease.---

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From time immemorial the Tonsils have been recognised as Structures, which in certain individuals and families called for the frequent attention of the Medical Practitioner .

## Follicular Tonsillitis. Angina lacunaris vel follicularis

It is not our intention to discuss the general symptoms, diagnosis &c of these changes, but rather to ascertain, as far as possible, the most recent views and opinions of their etiology and pathological changes.

On examining the throat a few hours after the rigor and rise of temperature associated with the commencement of follicular tonsillitis, it will be observed that the faucial tonsils are red and swollen, a few hours later whitish, elevated spots, make their appearance and these on closer inspection prove to be drops of exudation, issuing from the orificies of the crypts . These may be seized and pulled away, when they become drawn into threads, which retain an attachment to what is left in the crypts. Fresh exudation is formed and expels what is already present . The exudation finally gravitates downwards and becomes



coalesced with similar drops of exudation . Thus to a certain extent the Tonsils ~~get~~ covered with this whitish exudation, it is dense and adheres to the Tonsil . It is not however a pseudo membrane as it can be removed without injury to the surface on which it lies ,

Microscopically it consists mainly of leucocytes with a few epithelial cells , No. febrin is present according to Fraenkel (48). There are however large numbers of micro-organisms lying free and in the leucocytes mainly the streptococcus, though the Staphylococcus, Pneumococcus and the Bacillus coli communis are also found (Lemoine (42) and quite recently Nicolle and H<sup>e</sup>erbert (82) have demonstrated the presence of the bacillus of Freidlaender,

Microscopic examination of the Tonsil after removal in this condition, shows mainly a great exudation of leucocytes from the follicles- so great is the exudation that it almost conceals the epithelial cells. Fraenkel (48) considers "that the anatomical changes in lacunar Tonsillitis consists essentially in an enormously increased transudation of leucocytes, which cannot be due to the transudation being facilitated as there is nothing to be seen as far as the epithelium and the follicles are concerned to justify such a conclusion". He thinks therefore that we must suppose

that the influx of leucocytes to the follicles and adenoid tissues of the Tonsil is increased and therefore that angina lacunaris is a genuine inflammation of the parenchyma of the tonsil .

Sokolowski (51) views differ, in that he considers that the histological changes are more of a diphtheritis process, but of a less severe kind than that met with in genuine diphtheria . He found crypts distended and completely filled with a net-work of fibrin in which were large numbers of lymphoid cells and micro-organisms , In all cases fibrilles were found. A very few necrotic patches and these were only of the superficial layers of the tissue . The adenoid tissue itself was normal with the exception of an increased infiltration .

It was Stohr(5) who first realised that the destruction of the tonsillar epithelium by the emigrating leucocytes might permit the entrance of infectious materials into the tissues and that the very means taken to ward off the attacks of micro-organisms from one part of the Body (the Tonsils) might, under certain circumstances, allow pathogenic microbes or their products more quickly and easily to enter the circulation.

Fraenkel (48) points out that the idea is probable that an immigration takes place with relative difficulty on the same road as an emigration, for immigrating

microbes advancing would be met by the emigrating stream of leucocytes; circumstances which would make it very difficult for the advance of the micro-organisms, and he goes on to say that one is able by Grams' method to prove that in the Tonsils affected with lacunar tonsillitis the presence of micro-organisms can be made out even within the follicles, but MacIntyre (49) and others hold that no form of pathogenic micro-organism is to be found detected during this acute affection of the Tonsils which may not be present in the normal buccal cavity .

The bacterial examination of the Tonsils and fauces in such conditions is apt to be fallacious, owing to contamination with the organisms in the buccal secretions and Lemoine (42) has adopted the following method of isolating them in a large number of cases . The patients were made to cleanse their mouths as far as possible with sterilised water and after this the surface of the Tonsils was swabbed and dried with sterile cotton wool ; a minute spot was cauterized with heated pipette and through the centre of the small eschar resulting a capillary tube was introduced and a minute quantity of fluid obtained from which cultures were made .

Hofmeister, F, (56) and others have shown that the mitotic divisions of leucocytes to be observed in

adenoid tissue are far more numerous in animals after meals, and when they are in good health, than when they are debilitated. Looking at it from an everyday point of view medical men, as far as lies in their power, go to see infectious cases when they are not feeling 'fagged' and generally shortly after meals, as it is known that our liability to infection is not so great as when we are fagged and require food. Again we know that those individuals who are below 'par' - if we may use the term, are more liable to attacks of angina follicularis - as those who are overworked in Hospitals and young women at or about the menstrual period.

So, in the Tonsil then, as long as the outward stream of leucocytes is active (i.e. when the individual is in good health) micro-organisms are not likely to gain a footing.

We have seen that the leucocytes may do one of three things:-

- I. That they may pass per the lymphatics into the circulation.
- II They may remain as Stationary cells.
- III May make their way through the epithelium into the crypts.

why then, if as Gulland imagines, there is sometimes a call for leucocytes in other situations, and that those



leucocytes which would normally have passed through the epithelium, into the crypts, pass into the circulation, when the tonsil is attacked by micro-organisms should not ~~more~~ <sup>those</sup> leucocytes, which would normally have passed into the circulation, pass through the epithelium and thus lead to the increased transudation. But at present we cannot go further than this, and any views at all on the subject must be merely theoretical.

We know that the amoeboid cell with its higher power of resistance is able to withstand the attacks of micro-organisms and their products for a considerable time. In fact if the micro-organisms are few in number, it is now well recognised that the cells are able to destroy them, so that no further trace of the effect of the action of the micro-organisms can be distinguished; even when they (the amoeboid cells) succumb, other cells may take them up so rapidly and still retain their amoeboid powers for so long that the micro-organisms may be carried a considerable distance - as Woodhead (28) points out - from the original lesion, and may be deposited in a gland (as one of the cervical) in which active processes may have been so far diminished that the micro-organisms are able to multiply and thus the amoeboid cells actually afford supplies for the enemy in their own country, and so set up secondary mischief; if on the other hand the cells in the gland

are healthy and their vitality good they assist in destroying and disintegrating the micro-organisms (thus we see how common it is to have the cervical chain of lymph glands affected and enlarged following upon attacks of lacunar Tonsillitis .

What are then the pre-disposing causes of Angina lacunaris?

Fraenkel (ibid) reminds us that it frequently follows intra nasal operations , especially when the galvano-cautery is used and he remarks that he has never been able to distinguish any difference between the spontaneous and the traumatic variety . He does not think the hypothesis is probable "that the excitors of inflammation<sup>in</sup> make their way into the Tonsils by the same route by which the leucocytes make their way out" but "supposes the injury which damages the protective epithelial lining of the nasal cavities throws open the doors to the excitors of inflammation and that through this door they obtain access to the Tonsils from within, by way of the lymph stream", but here we are confronted with Gullands' statement , that the Tonsil is a peripheral lymph gland and has no lymphatics leading to it .

Colds, damp, surface chills, and certain diathesis

have at least a pre-disposing effect and even families exhibit similar tendencies . It is more common in the young, especially when there is existing hypertrophy.

All that can at present be said is that the organisms or their products have some casual relation to the condition and we must hope in time to be able to classify these inflammations of the Tonsil, under their respective organism or general condition which arises therefrom .

For we know that although a septic process may begin and end in the Tonsils, it is sometimes followed by a general infectious disease , or the septic process in the Tonsil may open the way for other microbes, as Löffler(29) reports two cases of diphtheria , in which the heart, liver, spleen, and kidneys, after death, shewed numerous strepto-cocci, Fraenkel(58) a case of diphtheria followed by ulcerative endocarditis, (Charrin(43) and others), and there are numerous cases on record where in this condition the tubercle bacillus has got a footing in the Tonsil, though this is still denied by some , as we shall see later . Further the so-called rheumatic affections of the joints so commonly associated are possibly, directly or indirectly, the result of an organismal action , though this question may be considered to be still sub judice.

At the British Medical Association Meeting (Laryngeal Section) 1895, there was a discussion on the question of the infectious nature of Lacunar Tonsillitis at which it was pointed out by MacIntyre that the clinical evidences are not confined to the local symptoms and signs at the seat of infection, that we sometimes have secondary infection of the respiratory, and gastro intestinal tract, that secondary mischief may be detected in the neck, (cervical glands) mediastinum, of the presence of albumenuria, which has been pointed out by Bouchard (59), Berkley Hill (60), and others, Alterations in the normal cardiac sounds, enlargement of the spleen, skin eruptions, pleurisy &c and irritation of such distant organs as the ovary and testes, all accompanied by distinct alteration in temperature, prostration and general manifestation of a severe constitutional fever.

Further the local conditions are frequently not obliterated after an attack of the disease and recurrent attacks may produce accumulative effects in the regions of the crypts, giving rise to the condition described by Ball (61) as "chronic lacunar Tonsillitis".

Again, climate-seasons-and situation- for we have such epidemics as those described by Stuart Tidey (46) occurring at Pontresina (known as Pontresina Throat)



among the summer visitors at the various Hotels and he believes that the disease is transferable from person to person ,and puts it down to the insanitary conditions and over-crowding in the various Hotels- must be taken into consideration .

The British Medical Association 1895 came to the following conclusions:-

- I. The clinical phenomena correspond in every particular with those of an infective disease .
- II . Cases have been cited in which the disease has undoubtedly been transmitted from one person to another .
- III. Various species of cocci and bacilli are to be found within the lacunae within the epithelial cells of Tonsil removed during the acute stage. Leucocytes in large numbers are found associated with those microbes .

There can be no doubt,as we have already seen,of the presence in the inflamed Tonsil,of great numbers of microbes. Some are of kinds which usually do,and others which do not,produce noxious effects upon their hosts. But it must be remembered that the products of microbie growths vary very much under differing conditions of their growths .

The abundant lymphatic tissue in the Tonsil renders the absorption of such products easy ,according to Wade(52). The completeness with which the tissue is able further to decompose and recombine the absorbed bye products ,or the incompleteness of these processes probably determines the extent to which the body suffers when the bye products ,if noxious,are absorbed.

Beyond this we cannot go as at present our knowledge does not permit it .

What has been said concerning the faucial Tonsils is also true of the Pharyngeal and lingual though perhaps not to so great an extent .

We have not as yet discussed the suppurative variety of Lacunar Tonsillitis -Quinsey which is probably a more aggravated condition , in which the micro-organisms have made their way further into the interior and the leucocytes being unable to cope with them,abscess formation has resulted .

The Text Books describe Quinsey as an inflammation more deeply seated affecting the stroma which tends to go on to suppuration . It may be peritonsillar and associated with cellulitis in the neck ,a condition described by Ludwig and usually termed Angina Ludovici .

Chronic Lacunar Tonsillitis: "Caseous Tonsillitis"

must not be confounded with hypertrophy of the Tonsil though in most cases the Tonsils are hypertrophied, but this is not invariably so . The disease is most common in young adults ,and seldom seen in Children.

On examining the Throat the orifices of the lacunae are occupied by cheesy masses ,which are retained in the crypt .

Aetiology: .The essential cause is some narrowing of the lacunar orifice,which impedes an abnormally free desquamation of the epithelial cells which is also present . Sokolowski(1 c)describes a sort of villous ingrowth of the epithelium into the lumen of the crypt,each minute villus containing a lymph follicle. Inflammatory processes on the surface of the Tonsils not infrequently lead to adhesions of the opposed edges of the lacunar slits ,causing a bridging over,with a partial,or even complete,occlusion of the lacunar orifice .

The cheesy masses are said by Ball(61)to be composed of epithelial cells in layers, a certain proportion of leucocytes ,fatty particles,cholesterine leptottrix filaments and various other micro-organisms, are also found in abundance .

## HYPERTROPHY of the TONSILS

sometimes designated, "Chronic Tonsillitis".

Faucial Tonsils:- All the several Tissues may be involved though in some cases it is chiefly the lymphoid elements without much development of the stroma. In other cases the fibrous matrix is increased and the organ is then harder, smaller, firmer and is cut with much greater difficulty. Mc Bride and Logan Turner (65) found a marked thickening of the epithelial covering and in one or two instances this was excessive. There is however no alteration in the type of epithelium.

Hypertrophy of the Tonsils, may, it is considered, be sometimes congenital, affecting both the pharyngeal and faucial tonsils.

Pharyngeal Tonsil:, may be one enlarged mass, or a number of vegetations, which have given rise to the term adenoids.

According to Trautmann (66) the pharyngeal tonsil may be divided into two parts; an anterior and posterior,

The central cleft, the "recessus medius", which divides the tonsil into anterior and posterior parts may be partly converted into a Canal by union of the ridges which bound it on each side.

Microscopically, it has been observed that the



epithelium covering the adenoids is also thickened and that the epithelium has changed its character, i.e., the ciliated epithelium with which that part of the pharynx is covered has disappeared and is replaced by ordinary squamous (Mc Bride and Logan Turner(65), Gottstein(68),

There is also a considerable difference in the fibrous tissue, in some cases a tremendous over-growth seems to take place in the neighbourhood of the blood vessels, described by Mc Bride and Turner, as a perivascular sclerosis, and they go on to show that it is due to the fibrous tissue<sup>formation</sup>, that gradual shrinking of the adenoid mass occurs. Brindel(69) points out that this has nothing to do with puberty and that shrinkage of Tonsil may occur at any age.

Aetiology, cold, damp, or what is known as "strumous diathesis" and heredity, are considered the chief factors in the production of hypertrophy.

Meyer of Copenhagen, in a paper published in 1895 (62) has collected numerous statistics from 'China to Peru' in which he was able to conclude that this hypertrophy occurs in Europe Asia and America in varying frequency; a warm climate appearing to favour adenoid growths less than a cold one- as it is common among the Esquimaux, though not unheard of in the tropics. Cold is then probably a factor in the production of hypertrophy by producing naso-pharyngeal catarrh, which

produce transient enlargement of the Tonsils . More especially we would notice the naso-pharyngeal catarrh associated with Measles and Scarlatina .

Killian(l.c)as we have already shown ,pointed out that in Animals with wide nostrils-as Man-who live much in closed rooms with many corpuscular elements in the air,we might expect the Tonsils to be enlarged and vice versa. Spicer(l,c)considers that the constant changes from one temperature to another occurring in modern civilised life (also among the Esquimaux-their Snow Houses have no outlet for fresh air or smoke except by the long tunnel of entrance and their Snow Huts are thus so close and smoky as to compel them to remove the greater part of their clothing )cause a corresponding activity in the erectile tissue in the nose,which in time causes a chronic irritability followed by a chronic congestion and discharge of a secretion differing from the normal which passes backwards and irritates the pharyngeal adenoid tissues causing Hypertrophy . Ordinary dust,trade dust ,and faulty voice production may all have a hand in its production .

With regard to the faucial tonsils ,Mc Bride and Turner consider that the thickness of the epithelium is caused by the fact that the food bolus in its

passage downwards is a source of frequent irritation to the surface of the enlarged faucial tonsils and the further fact that the excess of epithelium is found upon the free surface of the Tonsil and at the external openings of the crypts and not in their deeper parts .

Brindel found that in the pharyngeal tonsil thickening occurred most frequently in the growths removed from the younger patients ; that the change occurs in the smaller naso-pharynx as a result of intermittent pressure .This intermittent pressure is caused chiefly by the elevation of the soft palate in the act of deglutition pressing the soft pliant mass upwards against the walls of the space ,and releasing it again when the act is completed .

Mc Bride and Turner think also that the loss of the cilia and thinning of the epithelium which occurs in some cases may arise in another way,as one of their specimens demonstrated:viz:- "In a transverse section three of the crypts are seen considerably distended, and filled with a material possessing no definite structure, probably mucus, and faintly stained with rubin and orange. The pressure exerted by the presence of this secretion in a crypt whose orifice has become closed has caused a loss of cilia and a thinning of the epithelial lining layer. All stages of the process are visible in one crypt . Upon one area both the cilia and the

epithelium preserve their natural appearance as yet unaffected by the pressure, while in other parts the cilia only are destroyed ; the epithelium too, shows a varying degree of destruction ,being represented here and there by a single layer of flattened cells lying upon the basement membrane " .

The faucial may, or may not, be, hypertrophied along with the pharyngeal and vice versa .

The Lingual Tonsil, appears to be affected more commonly in the young adult ,after the faucial and pharyngeal tonsils have commenced to atrophy. ,Lennox Brown(57) considers that alcohol condiments ,very hot and very cold fluids ,chronic dyspepsia, gout and rheumatism all play a part in the production of its hypertrophy .

In connection with the tuberculosis of the Tonsil we shall have frequently to refer to hypertrophy ,as to whether the hypertrophied tonsil is capable of carrying on the process of emigration of leucocytes and another important question, which naturally arises, as to whether hypertrophy itself, is not an evidence of the presence of tuberculosis .



AFFECTIONS of the TONSILS due to special  
Micro-organisms .

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TUBERCLE OF TONSIL

It was Virchow who taught us that the Tonsils were peculiarly exempt from tubercle .

Within the last few years however it has been shown that not only are they frequently the seat of tuberculosis but one of the chief avenues by which tubercle enters the system .

Woodhead(38)points out that when Pigs are fed on tuberculous material held in solution-especially if the solution contains fatty or viscid material- in a very large proportion of cases these animals become affected with tuberculosis of the Glands associated with the Tonsils and it has long been known to Veterinary Surgeons that swellings at the angle of the jaw often leading, <sup>to</sup> abscess, were not uncommon in the Pig ;and in Holland Germany and Denmark every animal is examined for enlarged glands for any indication of a tuberculous nature in the region of the Tonsils.

Further, Woodhead points out that in many cases the Tonsils are themselves little, if at all , affected; i.e. there are no naked eye or microscopic lesions

which would enable one to say that these organs are tuberculous but the glands in immediate connection with them contain distinct foci of tubercle in various stages of growth and degeneration ;and he is driven to the conclusion that this method of infection of the glands of the Neck through the Tonsils must be of comparatively frequent occurrence especially in Children living under insanitary conditions and subjected to various devitalising influences, and it is evident that the infective agent, if introduced from without, must have gone through the Tonsils, which in these cases have been unable to deal in the usual manner with the tubercle. bacilli , i.e. "the leucocytes have only been able to carry bacilli from the tonsil to the gland in its immediate neighbourhood and at this point have succumbed, their protoplasm having served as a nutrient medium for the bacilli ", which multiplying and being reinforced by their progeny have continued the invasion still further, with the result that a chain of lymphatics and lymph glands has ultimately become infected .

Ruge considers primary tuberculosis of tonsils probable and reports the following case:- "A Girl aged 19 , had for several years suffered from enlargement of the Tonsil, since 1894 the right tonsil had become much more enlarged; in April 1895 cervical

caries developed . The patient was otherwise strong and healthy in appearance . No evidence of tubercle elsewhere, no enlargement of cervical glands. Right Tonsil as large as a Hens'egg with irregular surface. A piece of Tonsil removed ,showed scarcely any loss of superficial epithelium; ,there were tubercles in its substance in all stages of development !

Its occurrence with tuberculosis elsewhere has been recognised by several Writers as Lermoyez ,Dmochowski, Dieulafoy, Woodhead and others.

Clinically it can seldom be demonstrated for ulceration is usually absent and the only symptom may be the enlargement of the Tonsil .

Further the microscopic examination shows it to exist in the Tonsils in two histological forms .

1. The ordinary typical form of turbercle, as in a case reported by Purvis Stewart (64) "the case seemed to be merely one of ordinary chronic enlargement of the Tonsils with pharyngeal adenoids, associated with post-scarlatinal otorrhoea and enlargement of cervical glands in a Child aged 10 . No history of tubercle in the family . The Tonsils on a naked eye examination in no-way differed in their appearance from ordinary chronically enlarged Tonsils .

Microscopically; Scattered through the substance of the lymphoid tissue were large numbers of tubercu-

lous giant cell systems. Apparently most numerous close beneath the epithelial surface of the Tonsil. Staining by Ziehl-Niellson and Grams' methods failed to demonstrate any tubercle bacilli in the sections. The adenoids on section were found to be free from tubercle .

We seldom ever find that the Tonsils themselves break down from the invasion of tubercle and Lublinski (80) states that the reason why they do not do so is on account of the great development of fibrous tissue for the presence of tubercle appears always to be associated with hypertrophy .

II. A form in which the tonsillar tissue exhibits the structure either of an ordinary inflammation or hypertrophy with neither giant nor epithelial cells , but in which according to Dmochowski (37) the bacilli can be stained in the tissues in enormous numbers , and are found in the lymph channels in the Tonsil and leading from the Tonsil to the cervical glands , in such numbers as to resemble a pure culture .

These views are supported by Lermoyez (40) in a paper read before the Societe Medicales des Hopitaux of Paris in July 1894. Dieulafoy (38) on hearing Lermoyez's paper undertook the same investigations and his results were such as to lead him to fully acquiesce in Lermoyez's views .



Dieulafoy considers that the tubercle bacilli found in the Tonsils give rise in many cases to a form of masked or sluggish tuberculosis, characterised by an increase of phagocytes and hypertrophy of the lymphoid tissues with subsequent involvement of the cervical glands and not by the formation of typical tubercles with giant and epithelial cells, thus agreeing with Dmochowski .

Dieulafoy obtained Tonsils from 61 Cases treated by tonsillotomy in the Parisian Hospitals, mainly the Hospital des enfants . With this material he inoculated Guinea Pigs, with the result that in eight Cases the Animals became tubercular. In the same way adenoid vegetations removed from thirty five cases were inoculated into Guinea Pigs and in seven instances the Animals became tubercular.

Cornil (63) explains the production of tuberculosis by inoculation in so large a proportion of Dieulafoys' cases by the assumption that the tubercle bacilli had accidentally become entangled in the follicles of the Tonsils and among the vegetations .

Pluder and Fisher (73) out of 32 Cases examined, five shewed evidence of tuberculosis .

Sclenker and Krückmann (77) found in post mortem material two cases of primary tubercle in the Tonsils.

Kruckman(79)in post mortems of 25 tubercular patients found the following:- in twelve tubercle was demonstrated in the Tonsils and in every case in which the lymph glands of the neck were infected .

Strassmann(75)found tubercle in the Tonsil in 13 out of 15 Cases .

Rugé examined microscopically Tonsils in 17 Cases mostly young adults, seven had evidence of tubercle elsewhere ,and he found tubercle bacilli present in 5 ; all having Pulmonary Tuberculosis .

Adenoids, There is some considerable divergence of opinion as to whether <sup>the</sup> pharyngeal tonsil is as liable to the invasion of the tubercle bacilli as the faucial.

Pluder and Fisher consider the hypertrophy of the pharyngeal tonsil is an independent affection ,which has nothing to do with true "scrofula"because the great dis-semination of the growth not only among the lower classes ,but as much, and even more, in the middle and well-to-do, its good prognosis , its rapid subsidence in consequence of treatment. Further the fact that Children with an undoubted scrofulous tendency can often escape a marked hypertrophy .

Trautmann(74) says that although he made careful search in Adenoids ,in the follicles, in the secretions, he neither found giant cells nor tubercle bacilli ; in spite of it he holds tubercle as the origin of the hypertrophy.

But however, the giant cells sought for in vain by Trautmann, were found by Pilliet in his histological preparations .

Broca(83) examined microscopically 100 adenoids without a single positive result .

Gottstein(68) out of 33 Cases of Adenoids found four infected .

Brindel (69) out of 64 examined found giant cells and caseation in eight ; only one examined bacteriologically and out of forty sections stained only three or four bacilli, near a nodule, were found in one section .

Lermoyez(72) reports two cases of tubercular adenoids.

Mc Bride and Turner(86) in 32 cases observed five had tuberculosis stamped in the tissue .

Cornil and Lermoyez (84) holds the proportion to be about one in seventeen.

It is probable that when tuberculosis occurs in the pharyngeal tonsil (if it does occur) it is usually associated with chronic nasal catarrh and in chronic nasal catarrh the warming, filtering and moistening action of the schneiderin membrane is very much interfered with and therefore the air is apt to arrive cold, dry and germ laden, in the delicate pulmonary alveoli (Freudenthal(85)).

Nicholl(55) is of the opinion that in a number of

cases chronic nasal catarrh may itself be tubercular and compares the chronic softening and suppurative ~~in connection~~ artheritis with ~~bone~~ which were formerly considered affection apart from tubercle and which now-a-days are as a matter of course pronounced tubercular and treated as such .

Mc Bride and Turner (86) examined the nasal mucous and nasal pharyngeal for tubercle bacilli , in one case in which Tonsils contained a few giant cells and tubercle bacilli but no caseation, with negative results .

Rugé considers:-

- I.. That the infection occurs by the blood as in military tuberculosis .
- II. By lymph as secondary to tuberculosis of glands, which he doubts .
- III. By the Sputum especially in tubercular phthisis ; in this he is supported by Kruckmann(79) and Strassmann(75) .
- IV. By the air, which he very much doubts .
- V. By food, especially milk and meat infected with bovine tuberculosis .

Tussan(44) considers that it is apt to occur where there is chronic irritation (as from tobacco and alcohol) which probably interferes with the resistance of these glands and the question naturally arises whether emigration  
ion



of leucocytes can go on while there is such chronic thickening of the epithelium covering the tonsils .

With regard to Adenoids Mc Bride and Turner(65) say "We "have not been able to satisfy ourselves that this "emigration of leucocytes takes place generally or in "any marked degree, from the pharyngeal tonsil, in the "hypertrophied states, where the thickened stratified "squamous epithelium, exists, nothing of the kind is "visible". Whether the same is to be said of the faucial tonsils is a question which has not as yet been answered. No- one so far has made any definite statement on the subject .

If we are to accept Dieulafoys' observations , it clears the road to a great extent , by accounting for the frequent occurrence of hypertrophy of the faucial and Pharyngeal adenoid tissue ; but at present, observations are so conflicting, whether it is because the methods of staining are imperfect, or because the whole tonsil has not been systematically examined -for a whole lobule might be healthy, while the neighbouring part may contain tubercle, and thus the condition pass unobserved - we are unable to say .

Mouret (81) reports a Case in which there was present , bacilli at the bottom of the crypts and

outside the tonsillar tissue, which would be sufficient to produce tubercle in an animal inoculated .

Thus we see that to prove the tonsil tubercular we must not rely upon inoculation alone, but on histological examination.

Where there is hypertrophy of the Tonsils associated with enlargement of the cervical glands there must probably, will tubercle, in one form or another, be found; where there is hypertrophy without enlargement or swelling of cervical glands, then the presence of tubercle, in any form, in the adenoid tissue must be considered very doubtful .

At any rate we are bound to recognise the fact that the Tonsils, faucial, and pharyngeal, are frequently the gates of infection for tubercle bacilli and these bacilli may, or may not, be allowed by the leucocytes to remain in the Tonsil or they may be carried by them to neighbouring Glands and thus infect them .

AFFECTIONS OF THE TONSILS due to the  
KLEBS -LOEFFLER BACILLUS

Everything points to the fact that diphtheria is a constitutional fever whose virulence is due to the toxic effects produced by the Klebs-Loeffler Bacillus.

Spronck(88)points out that although a bacteriological diagnosis of diphtheria is now very easily and certainly arrived at, nevertheless cases exist in which bacteriologists disagree. He points out that there are three varieties of diphtheria bacillus and these are differentiated by their size. The short bacillus, which closely resembles the pseudo-diphtheria bacillus of Von Hoffmann. The long bacillus is the most toxic of the three; the medium less so; and the small, least of all . Spronck considers that Von Hoffmanns' bacillus is probably derived sometimes from the true diphtheria bacillus, at other times from a slightly virulent pseudo-diphtheria bacillus .

Hennig of Königsberg(89)at the recent Congress at Wiesbaden ,stated that he had made examination of 63 cases in relation to Loefflers' bacillus which he had found present in thirty five cases and in some only of these was the disease clinically true diphtheria; in others it was a simple tonsillitis . Moreover the bacillus was not present in seven cases that subsequently

exhibited typical paralysis and he therefore concludes that this microbe cannot be invariably the determining Agent in ~~diphtheria~~, and that no treatment based on the hypothesis that it is so can be accepted as specific.

. But so very much has lately been written on the subject of ~~diphtheria~~, especially since the introduction of the treatment by its antitoxin that to go fully into the subject must be considered as beyond the scope of this Thesis .



AFFECTION of the TONSIL in connection with  
SCARLATINA

We know that in connection with Scarlatina, sore throat is of frequent occurrence. It usually proceeds the appearance of the rash and consists of a yellowish exudation on the fauces and tonsils. In later stages suppuration may follow; the nasal mucous membrane also inflames and there is much mucous secretion and very frequently the sub-maxillary and cervical become enlarged and tender. Diphtheria is often also associated with Scarlatina. In a number of cases Diphtheria bacillus has been found late in the disease while in others only the streptococcus has been found.

Ranke(90)notes that out of 142 Cases of Scarlatina admitted into Hospital, of these 92, or 64.7% , had exudation on the Tonsils ; 89 of these were examined bacteriologically .

He places them under the following groups:-

1. Cases admitted with Scarlet fever and diphtheria and examined at once .

There were 67 such cases. Streptococci found alone in 38.8%, the Streptococci and diphtheria bacilli in 47.7% and the diphtheria bacilli alone in 5.9% ; in all the diphtheria bacilli was present in over 53% ; as

compared with true diphtheria the diphtheria bacillus being found in 91.6%. Thus the bacterial examination in scarlatinal diphtheria gives different results to that of primary diphtheria . He considers that the simplest explanation is that the scarlatinal angina pre-disposes to the diphtheria infection .

II. Cases admitted with Scarlet Fever but in which diphtheria subsequently developed .

Here there were ten cases. In one diphtheria found in pure culture. In seven diphtheria bacilli and streptococci . In two streptococci alone .

III. Cases of primary diphtheria in which Scarlet fever developed later .

Here there were twelve cases. The bacterial examination resembled that in true diphtheria. He considers that the Scarlet fever was primary in these cases and that the diphtheria developed during the incubation period of the Scarlet fever .

IV. Cases in which the Scarlet fever and diphtheria occurred in patients in Hospital for other diseases. In two the streptococci alone were present.

Goodall(91) in a paper on the pathology and eitiology of post-scarlatinal and post-morbiliary diphtheria, makes the following statement (which is rather opposed to that of Ranke) "since he had employed the bacteriological tests, he had abandoned the opinion he once held as to

diphtheritic character of the majority of cases of anginal scarlatina, though the bacilli were to be found in a considerable number of cases of typical scarlatina, and of other diseases, as Measles or Pneumonia.

The cases of Scarlatina in which the diphtheria bacilli occurred were mostly of a mild type and their mixed character could not have been suspected from a clinical examination only.

In the Eastern Hospital from 1892 to 1894 there were 83 Cases of Measles admitted. In 17 (or 20.4%) the diphtheria bacilli was found.

Notwithstanding the fact that we do not yet know the specific organism which is the cause of Scarlet fever, the frequent presence of streptococci must be noticed along with that of the diphtheria bacilli.

The streptococci has been found in the Throat, Glands, Kidneys, Ear discharge, Valvular Vegetations &c.

On these grounds Marmorek (92) injected anti-streptococcic serum in 96 Cases of Scarlet fever at the Trousseau Hospital.

Of these, five died, four from diphtheria and one from pneumonia.

The most marked effect of the serum was on the swollen glands, which subsided so rapidly that there was no suppuration in a single case. In the event of albuminuria one or two injections caused its disappearance.

Not only did the serum seem to prevent grave complications ,but it also caused the rapid disappearance of false membrane from the throat; the general state of the patient rapidly improved. The only bad effects observed were transient erythemas .

Marmorek,while admitting that the series was too small to warrant any definite conclusion,is still of opinion that the serum treatment is of considerable use in reducing the severity of the attack .

Therefore until we are able to assign to Scarlet fever a specific organism of its own we can only treat the tonsil condition as one of an infective angina lacunaris .



TONSILLITIS and its connection with  
acute RHEUMATISM

Tonsillitis occurs so frequently in direct and immediate association with the articular symptoms of rheumatism that the pathological connection cannot be doubted .

Wade(52)observed that" inasmuch as it so frequently follows an infective disease ,and that all its features can be explained by the assumption of microbic infection there is a high degree of probability that it is an infective or microbic disease ". As yet no special bacillus has been assigned to rheumatism and since not all cases of tonsillitis are followed by rheumatic fever one or two things are pretty certain "either that a special bacillus,or group,must,to produce rheumatic fever,be added to those which cause tonsillitis; or that it requires a special constitution of the system(a'rheumatic diathesis')to allow of the development of acute tonsillitis and the rheumatic fever which follows it ,but with a considerable gap between the two,the gap being bridged over by a continuous or remittent catarrh". Wade suggests that it is a mixed poison in connection with the'rheumatic diathesis' and that these mixed poisons are the products of

microbic growth.

Lees(54)holds that in many cases of rheumatism more or less 'Sore Throat' exists and "suggests that a microbic focus existed in this locality and developed toxins which poisoned the patient ". The poison may be indefinitely detained in some part, possibly the closed follicles of the Throat".

Jaccoud,(93)notes that it is a striking fact that the organisms found in the Tonsils are exactly the same as those found in the tissues which are the seat of the lesion and he further remarks that in fact the tonsils or any tissue shewing a lesion ,may allow the organism to enter,and refers to a case in which a wound of the foot seemed to be the lesion to blame .

As we are unable to prove these suggestions in the stringent manner required by Koch let us look at them from a clinical standpoint .

We know that very frequently tonsillitis occurs either before,during,or between attacks of Rheumatic fever;that it may affect all three tonsils,though the faucial most frequently . Clinically it cannot be distinguished from ordinary tonsillitis except that it will not give way to ordinary treatment-but to anti-rheumatic !

There may be frequent attacks of tonsillitis and then an attack along with rheumatic fever. Again

there may be tonsillitis in a 'rheumatic' subject along with cardiac symptoms but without Arthritis, as in a case reported by Havilland Hall (94) and another by Mc Donnell (95) (though there is no proof in the case reported by the latter that the conditions were rheumatic at all ).

Wade (52) makes the following classification:-

- I. Tonsillitis with or without abscess, which is neither, preceded, attended nor followed by rheumatism.
- II. Repeated attacks of Tonsillitis without Rheumatism, then one immediately followed by Rheumatism.
- III. Cases of repeated attacks of Tonsillitis without Rheumatism, then an attack remotely followed by Rheumatism .
- IV. Cases of first attack immediately followed by Rheumatism and to which might be added:-  
Attacks of Rheumatic fever remotely followed by Tonsillitis, (Cheadle (53) .

When we turn to the treatment of these Cases of Tonsillitis it is then that the clinical evidence becomes so important , for all are agreed that the most satisfactory treatment, in fact the only treatment of any avail is that of Salicylates; but till we get further evidence , more especially pathological, the connection between tonsillitis and rheumatic fever must be considered still undetermined .

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## CONCLUDING REMARKS

Purposely no reference has been made to Syphilis, malignant disease, and such conditions as Varix etc, as these subjects are somewhat outside the limits which were intended in this Thesis .

The role played by the Tonsils as a point of entrance of germs into the system is now generally realised.

Gerhardt (96) has termed the Tonsils a physiological wound, an inlet guarded however by leucocytes which protect the system against the entrance of these germs, and it has been seen that if from any reason the energy of the leucocytes is diminished, or the Tonsil in an unhealthy condition (as hypertrophy) then the germs of disease gain access to the system .

We have followed the development; discussed the anatomy and functions of the tonsils, and subsequently endeavoured to arrange as far as possible, with our present knowledge, the various affections of the Tonsil under their causal micro-organisms or their supposed toxic products .

I am conscious of having omitted much of importance but the subject turned out a larger one than I at first supposed and the abundance of material rendered



the task a more difficult one.

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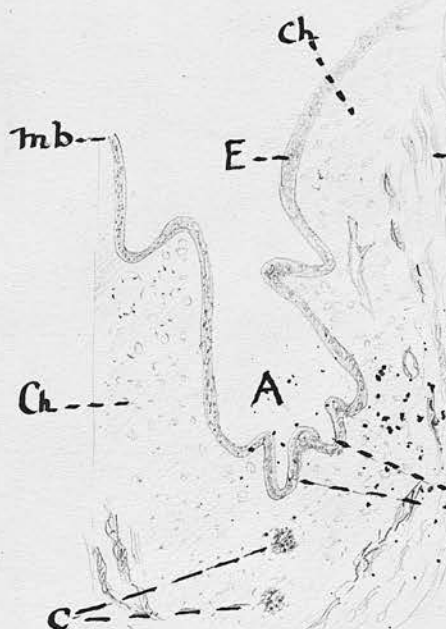
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Somerset.

April. 1897:

Trans. Section through the developing Tonsil in a human embryo, at 4<sup>th</sup> month of gestation. (Retterer.)



A Resulting tonsillar fossa from the involution of primary palatine epithelium.

B. Secondary involutions in section.

C. Cross section of buds.

E. Palatine Epithelium.

Mb. which is continued over the involutions

Ch. chorion

Tm. Muscular fibres.

Fig. I.

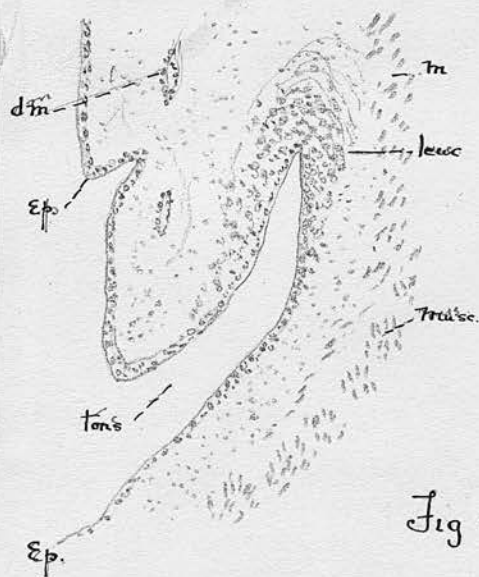


Fig. II

Transverse Section through pharynx of a rabbit foetus (26 days). Note condensation round crypt

(Gulland)

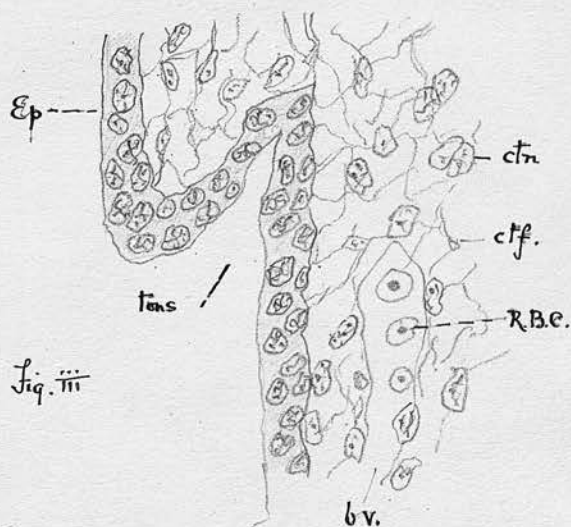
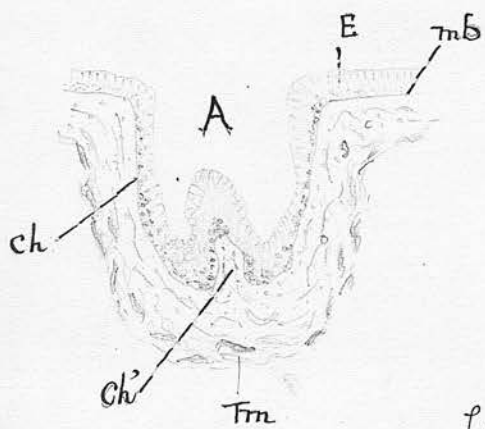


Fig. III

Trans. sect<sup>n</sup> through tonsil of rabbit foetus (12 days)

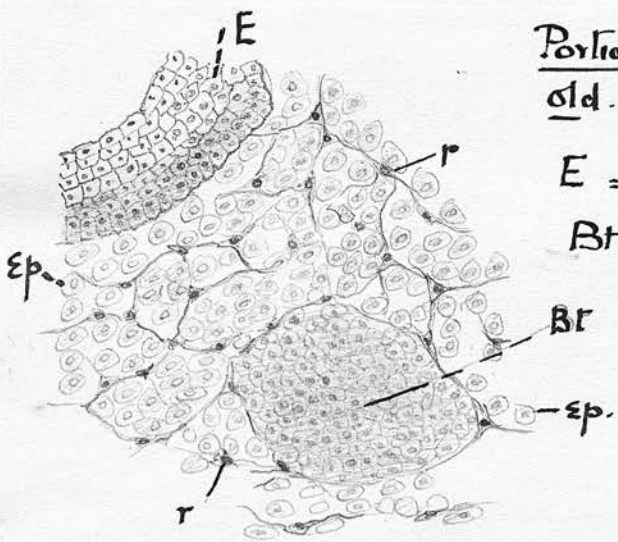
Gly. Hartnack q. wat imm (Gulland).



Longitudinal Section of tonsillar region in the foetus of a sheep. (Retterer.)

- A. Tonsillar Fossa.
- E. Epithelium
- ch. Chorion.
- mb. basilar Membrane.
- Tm, Muscular coat.

Fig IV.



Portion of the lobe of a tonsil <sup>of the calf,</sup> three or four weeks old. Magnified  $\frac{160}{1}$ . (Retterer.)

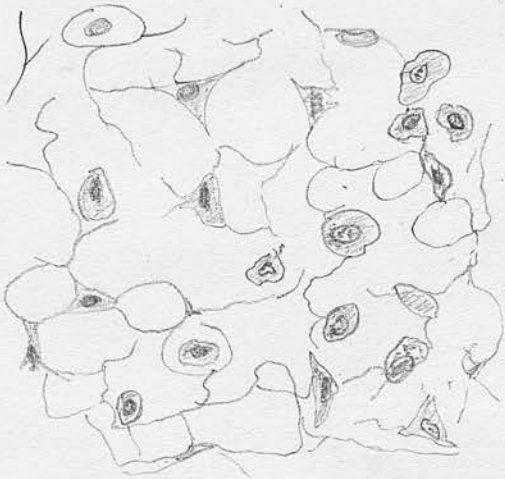
- E = Epithelium of diverticulum
- BT = Bud terminal with surrounding Connective tissue etc.
- Ep = Epithelial cells.

Fig. V.

Fig. VI.



A drawing from a section of a normal tonsil from which the cells have been removed, showing the reticulum of two nodules. (Hodgkynyl).



High power drawing, showing the reticular tissue of germ-center or nodule. (Hodgkynyl).

Fig. VII.



Fig. 8.



The Epithelium covering the tonsil, showing emigration of lymphoid cells, & the process of rarefaction described by Hodeupyl. (Hodeupyl).

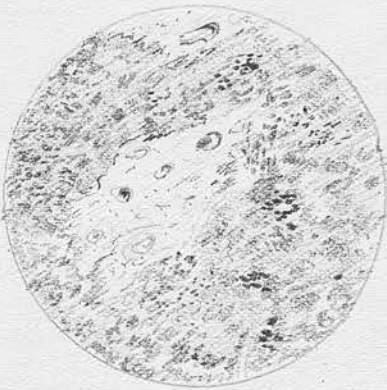


Fig 9.

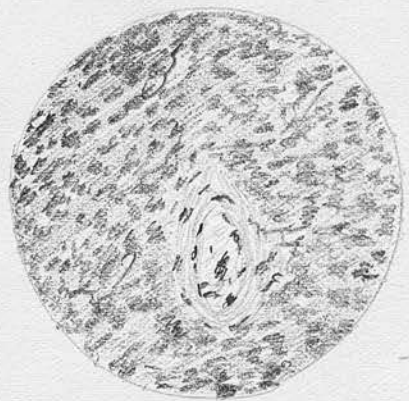


Fig 10.

(McBride and Turner).

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